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THE EFFECT OF X-RAYS UPON DOMINANT MUTATION IN MAIZE[1]

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No dominant mutations have been found in x-ray progenies of barley and maize, in the course of experiments in which many hundreds of recessive mutations have been detected. Many dominant alterations appear in the first generation progeny produced by the use of x-rayed pollen, but these alterations are lethal to the gametophyte and are therefore not reproduced in later generations. The recessive mutations detected are at least partially haplo-viable, since they must be transmitted through both male and female gametophytes before their effects may be observed in homozygous individuals. Since all of the induced alterations which are viable in haplophase are wholly recessive, these experiments provide no evidence of the production by x-rays of any mutant gene of positive action. The induced mutations observed may be merely gene losses tolerated by the gametophyte.[2,3]

The evidence against the occurrence of x-ray-induced dominant mutation is, however, inconclusive, for the following reasons:

1. The number of genes capable of showing the effects of dominant mutation may be much smaller than the number capable of showing recessive mutation, since many genes may be fixed by natural selection at a level maximal for phenotypic effect. The preponderance of recessive mutations may be only a reflection of the preponderance of such alleles in the genotype of the irradiated individual. The possibility of inducing dominant mutation therefore may be tested critically only by the effects upon known recessives, that is, genes with known dominant alleles.

2. Among known recessives many may be themselves deficiencies and therefore incapable of mutation. Critical evidence of failure to mutate to a dominant allele therefore may be obtained only from recessive genes which have previously been known to mutate to dominant alleles.

3. The only recessive alleles which meet this requirement are the so-

called variegation genes, which may be regarded as unstable recessives mutating frequently to a dominant allele. Each of these is characterized by a high spontaneous frequency of dominant mutation, so high that the effect of x-ray treatment in inducing additional dominant mutations probably would be inappreciable. Demerec's experiment[4] with the unstable miniature-3 of *Drosophila virilis* is an example of this difficulty.

It is possible to avoid these difficulties in the case of one gene. The recessive gene a_1 in maize has several known dominant alleles. Rhoades[5,6] has shown that in the presence of another gene, *Dt*, mutations of *a* to various dominant alleles occur. With increasing dosage of *Dt* the frequency of dominant mutation of *a* is sharply increased, and in endosperms triplex for *Dt* and for *a* (*a a a Dt Dt Dt*) more than 100 mutations per seed are usually found. In the absence of *Dt* no dominant mutations of *a* have been reported, though *a a a dt dt dt* endosperms have been closely examined in large numbers. (Dr. Rhoades informs me that he has seen one instance of an apparent "dot" of anthocyanin colored tissue in the aleurone cells of an *a a a dt dt dt* seed.)

The gene *a*, then, is capable of mutation to a recognizable dominant allele, and can be tested for response to x-ray treatment in genotypes in which spontaneous dominant mutations of the gene rarely if ever occur. Since the effects of mutation are clearly recognizable in minute sectors of tissue, the treatment may be applied at a fairly advanced stage in endosperm development, so that many hundreds of cells may be tested for mutation by examination of a single endosperm. It is possible therefore to test for the occurrence of this mutation in practically unlimited populations.

The experiment was conducted as follows: The seed to be irradiated was produced by the cross *a a* × *A a*, both parent stocks being homozygous for *dt dt* and for the complementary factors required for aleurone color development. The endosperms of half of the seeds produced are *A a a* (colored aleurone). These serve to indicate the size of the sectors resulting from genetic alterations induced by irradiation at the stage of development chosen, since induced deficiencies or recessive mutations of *A* in these endosperms result in sectors deficient in aleurone color. In the colorless seeds (*a a a*) induced dominant mutation of any one of the *a* genes would result in a corresponding sector of colored aleurone. The colored seeds thus provide a basis for estimating the number of opportunities for the occurrence of detectable mutation in the colorless seeds, and for comparison of the relative frequency of induced dominant mutation and induced loss of *A* due to recessive mutation or deficiency.

X-ray treatment was applied to the ears 73–81 hours after pollination, following preliminary experiments which indicated that treatment at this stage results in sectors of suitable size. Treatments were made on field-grown plants by means of a mobile x-ray unit with self-rectifying tube,

operated at 88 K.V.P., 4.5 ma. tube current, and 15 inches target distance, with no filtration other than that of the overlying tissue. Under these conditions the time of treatment is 2.48 minutes per 100 r. The doses applied, uncorrected for filtration, were 800 r and 1600 r.

The lower dose was applied to 21 ears, which yielded a total of 8003 seeds. The effect of the treatment was clearly greater on the exposed side of the ear than on the opposite side, as indicated by the frequency of deficiency sectors and also by the occurrence of "scarred" endosperms. There was also appreciable variation in dosage along the long axis.

The average size and frequency of sectors showing "*A*-loss" (that is, loss of the *A* phenotype), was estimated by detailed examination of a small sample of seeds. From each of six representative ears given the lower dose of radiation, three seeds were taken from the treated side and three from the opposite side, with approximately even distribution along the long axis of the ear. Each of the 36 seeds was closely examined under magnification by the use of a dissecting microscope. This permits determination of the size of sectors by direct count of aleurone cells affected. In small sectors due to loss of *A* the aleurone cells are not wholly colorless, but the contrast in color is sharp enough to make the outline of the sector quite definite. In large sectors, produced by irradiation of the ear about 30 hours after pollination, the aleurone cells of sectors resulting from loss of *A* are colorless except for a rim of faintly colored cells about the margin of the sector. There are in addition occasional sectors of dilute aleurone color, probably resulting from similar changes affecting an unknown gene or genes. These are readily distinguishable from sectors involving *A* in the early-treated material, but are less distinct when the sectors are so small that marginal color may cover the entire sector. In order to minimize the error from this source, all sectors which seemed possibly due to loss of genes other than *A* were omitted. These made up nearly one-third of the aleurone sectors observed on the treated seeds. The ratio of dilute sectors to colorless sectors on early-treated seeds of the same cross is considerably less than 1:2, and it is therefore probable that the actual frequency of *A*-loss in the experimental material is somewhat higher than the data below would indicate. The observations are summarized in table 1.

The 18 seeds representing the exposed side of the treated ears (seeds Nos. 1 to 3 on each ear) yielded 618 sectors, or an average of 34 per seed; while those representing the opposite side yielded 248, or an average of 14 per seed. The sectors varied rather widely in size, the average number of aleurone cells included being 112. The frequency of *A*-loss sectors on untreated ears, determined by similar examination of 10 representative seeds from each of the 19 control ears, was 0.12 per seed. The 23 sectors found ranged in size from 9 to 750 cells.

The approximate number of aleurone cells in the entire endosperm was

determined similarly by counts of aleurone cells in representative areas. The number of aleurone cells in seeds of average size was found to be about 160,000. It may be roughly estimated, therefore, that at the time of treatment the number of cells per seed whose genetic alteration could be detected by sectors in the mature aleurone is about 1400 (160,000/112).

The colorless seeds of the same treated ears permit the detection of mutations of *a* to any one of the various *A* alleles producing aleurone color. Each endosperm cell contains three *a*'s, each of which is capable, under the influence of *Dt*, of mutating to a colored aleurone allele. If the phenotypic effects of mutation and of deficiency are shown by the total cell progeny of the irradiated cell, or by the same fraction of this progeny, the result of mutation should be a colored sector equal in area to the color-deficient sectors produced by deficiency.

If either alteration requires for its realization a period of one or more cell generations in excess of the other, the resulting sectors may be of unequal size. It is not to be expected, however, that delay in realization will regularly reduce the size of sector by one-half for each cell generation. The sectors observed include only the cells of the aleurone layer which are included in the block of tissue affected; their reduction in size with advancing endosperm development depends upon the pattern of development of the tissues concerned. Actually, the average size of deficiency sector observed in ears treated at periods after pollination ranging from 73 to 81 hours was not changed enough to show any consistent relation among the 21 ears treated. Ears treated 96 hours after pollination yielded smaller but readily recognizable sectors. It is evident, therefore, that even if there were a delay in realization of gene mutation for several cell generations after irradiation, the resultant sectors would be detectable. Colored sectors as small as 10 cells in size are clearly recognizable on close examination of the seeds without magnification, and a single colored aleurone cell on a colorless seed is readily identified under magnification.

All of the colorless seeds were closely examined without magnification for sectors of colored aleurone. Occasional discolored areas of pericarp or endosperm were found, but under magnification these were readily distinguishable from colored aleurone sectors. No sectors of colored aleurone were found, among the 3832 seeds examined. On the basis of 1400 cells per seed, each containing 3 *a* genes capable of dominant mutation, this represents approximately 16 million chances for detectable dominant mutation ($3832 \times 1400 \times 3$). The treatment to which these cells were exposed produced in the adjoining colored seeds approximately 100,000 losses of *A*. Since the number of *A*'s tested for frequency of *A*-loss was only one-third the number of *a*'s tested for frequency of dominant mutation, the experiment shows failure to induce dominant mutation in a trial on a scale sufficient to produce about 300,000 *A*-losses.

A similar examination was made of the ears irradiated at 1600 r. At this dose the number of *A*-losses on seeds on the exposed side of the ear is too great to permit an accurate count. On the opposite side of the ear the frequency is very high also, but samples representative of the central portion of this region (amounting to approximately one-sixth of the ear) were suitable for examination. In 8 of the ears treated a sample of 6 seeds per ear was examined, the sample in each case being taken from the two rows least exposed to the radiation. The number of *A*-losses from these samples averaged 20.3 per seed. This is about three times the frequency of *A*-

TABLE 1

FREQUENCY AND SIZE OF SECTORS DUE TO *A*-LOSS FOLLOWING X-RAY TREATMENT

Ear	No. 870	No. 876	No. 880	No. 881	No. 882	No. 884	Total
Interval, pollination to irradiation (hrs.)	75	75.5	80.5	81	81.5	75	
Number of sectors							
Seed No. 1	22	32	26	36	49	40	205
No. 2	30	31	38	33	34	29	195
No. 3	49	43	34	23	37	32	218
No. 4	3	9	13	14	6	15	60
No. 5	20	12	17	12	14	9	84
No. 6	11	16	13	18	24	22	104
Number of aleurone cells per sector (frequency distribution)							
1–10	12	12	16	9	23	18	90
11–50	38	43	48	42	47	41	259
51–200	71	64	46	63	82	69	395
201–500	14	23	27	22	11	19	116
500+	0	1	4	0	1	0	6

losses in seeds similarly located on the ears treated at 800 r, and inspection of seeds in other regions of the ears indicates that this is a fair estimate of the relative frequency of *A*-losses from the two doses used. The average size of sectors was 113 cells.

The 15 ears treated at 1600 r produced a total of 2487 colorless seeds. These were closely examined for sectors of colored aleurone, and in addition a sample of 50 seeds from the most heavily treated region of each was minutely examined under magnification. No sectors of colored aleurone were found, with the exception of a single doubtful spot possibly including 3 aleurone cells, which could not be positively identified. The frequency of *A*-losses, producible by the same dose in an equal population of *A*-genes, estimated as before, is about 600,000.

The susceptibility to dominant mutation of the *a* genes used in this experiment was tested by pollinating each of the parental stocks by a homozygous stock designated *a*-dl *Dt*. The gene *a*-dl is one of a number of "dotless" *a* alleles arising by mutation of standard *a* under the influence of *Dt*. It is indistinguishable from *a* in phenotypic effect, but differs in showing almost no dots due to dominant mutation in the presence of *Dt*. In endosperm triplex for *a*-dl and *Dt* an occasional small dot may be found, as in the case of the similar mutant a^s described by Rhoades, but the rate is negligible in comparison to that of standard *a*. In the endosperms of *a a a*-dl *dt dt Dt*, produced by the crosses under discussion, the dots observed may be ascribed almost wholly to mutation of one of the two *a*'s under the influence of the single dose of *Dt*. The mutation frequencies for x-ray action and *Dt* action are, of course, not directly comparable, since it is not possible to apply the two stimuli for comparable periods or to comparable populations of cells.

The number of dots per seed in endosperms of *a a a*-dl *dt dt Dt* produced by crosses on the female parental stock was 2.95, on the colorless seeds of the male parental stock 2.1. In each case the dots represent the number of mutations produced by two *a* genes in the presence of a single *Dt* gene. In the *a a a* seeds irradiated, therefore, the number of mutations to *A* which would be expected in the presence of one dose of *Dt* is about 4 per seed (2.95 + 2.1/2). Rhoades[4] has shown that the frequency of mutation of *a* to *A* is disproportionately increased by increased dosage of *Dt*, and that about 17 times as many mutations are produced in *Dt Dt Dt* seeds as in *Dt dt dt* seeds. The population of 6319 seeds of *a a a*, which failed to yield mutations to *A* under x-ray treatment, was therefore capable of yielding about 400,000 mutations to *A* under the influence of homozygous *Dt* (6319 × 4.0 × 17).

Summary.—X-ray treatment failed to induce mutation of *a* to *A* (or to any other colored aleurone allele), in populations which, with the x-ray doses applied, were capable of yielding about 900,000 losses of *A* by deficiency or by mutation to a colorless allele.

The *a* genes used in the experiment, when combined with *Dt*, yielded numerous mutations to *A* or other colored aleurone alleles. The population irradiated was large enough to have yielded, under the influence of homozygous *Dt*, about 400,000 of such mutations.

[1] Coöperative investigations of the Division of Cereal Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture, and Missouri Agricultural Experiment Station, University of Missouri. Missouri Agricultural Experiment Station Journal Series 940.

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GRAPHICAL METHOD OF FACTORING THE CORRELATION MATRIX

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Multiple factor analysis starts with a square matrix R of order $n \times n$ with cell entries r_{jk} which are experimentally determined. The matrix R shows the correlation of each variable with every other variable in the test battery. The correlation matrix is symmetric since $r_{jk} = r_{kj}$. Some students write unity in the diagonal cells and this is legitimate for some problems. More often the diagonals contain the communalities h_j^2 which are so chosen as to be consistent with the rank of the matrix as determined by the side entries. The factoring methods to be described can be applied for any set of diagonal entries.[1]

The first object of multiple factor analysis is to find a factor matrix F of order $n \times r$ where r is the rank of the correlation matrix R_{jk}. The factor matrix F must be written so as to satisfy the fundamental relation $FF' = R$. The correlation matrix R can be factored into a factor matrix F in many different ways and F is not here unique. The current geometrical interpretation of R is that its entries r_{jk} show the scalar products of pairs of test vectors J and K which are not necessarily of unit length unless this restriction is imposed by unit diagonals r_{jj}. Since the correlation coefficients are scalar products, it follows that no reference frame is implied by R. Each row j of the factor matrix F shows the entries a_{jm} which are the projections of the test vector J on a set of r orthogonal reference axes. The lack of uniqueness of F is geometrically interpreted in the free choice of an orthogonal reference frame for the test configuration of n test vectors J. The reference frame is explicitly defined in F relative to the test vectors but it is not defined in R.

One factorial solution for F is to locate the first reference axis so as to maximize the sum of squares of test vector projections. The second reference axis can be located by the same criterion applied to first factor correlation residuals, and so on until a set of r orthogonal axes have been determined which correspond to rank r of the correlation matrix. In the writer's original formulation of this problem, it was called the *principal axes solution*.[2] In the first paper on this problem a computational method was described for finding the principal axes solution F. It involved first factoring the correlation matrix into a factor matrix F with any arbitrary orthogonal reference frame and a subsequent orthogonal transformation representing a rigid rotation to the principal axes. That solution involved the characteristic equation of the matrix $F'F$ and determination of its latent

roots. The next year Hotelling described an iterative method of factoring the correlation matrix directly into the principal axes solution which he renamed *principal components*.[3] Since the iterative methods, as well as all other available methods, of obtaining the principal axes solution are very laborious, it is of interest to students of multiple factor analysis to find shorter methods.

Consider the first column of F for the principal axes solution. Its elements are a_{j1}. It is desired to find the values of a_{j1} so that the first factor shall account for a maximum of the variance in the correlation matrix R. Then

$$r_{jk} - a_{j1}a_{k1} = \rho_{jk}, \tag{1}$$

where a_{j1} and a_{k1} are the first factor saturations of tests j and k, and ρ_{jk} is the residual to be minimized. Squaring and summing for all correlation coefficients, we have

$$\sum_{j}\sum_{k} r_{jk} - 2\sum_{j}\sum_{k} a_{j1}a_{k1}r_{jk} + \sum_{j}\sum_{k} a_{j1}^2a_{k1}^2 = \sum_{j}\sum_{k} \rho_{jk}^2 \equiv z, \tag{2}$$

where the sum of the squares of the correlation residuals ρ_{jk} may be denoted z. The partial derivative with respect to a_{j1} is then

$$\frac{\partial z}{\partial a_{j1}} = -4\sum_{k} r_{jk}a_{k1} + 4a_{j1}\sum_{k} a_{k1}^2 \tag{3}$$

from which we get

$$a_{j1}\sum_{k} a_{k1}^2 = \sum_{k} r_{jk}a_{k1}. \tag{4}$$

Let the sum $\Sigma a^2 \equiv t$. The summation Σra can be written as a matrix product Ra. Then

$$Ra = at \tag{5}$$

where R is the given square matrix, a is a column vector, and t is a scalar, namely, the largest latent root. The iterative process consists in starting with a trial vector u so that

$$Ru = v\Sigma u^2 \tag{6}$$

where $(v\Sigma u^2)$, or numbers proportional to these, constitute the trial vector u for the next iteration. The process continues until $v\Sigma u^2$ becomes proportional to the trial values u at which time the values of v $(= u)$ are the desired values of a_{j1} and the first column of F is then determined. The number of iterations can be considerably reduced by starting with R^2, or R^4, or even R^8. While that looks effective in a text book example, it is not so inviting a procedure when we are confronted with a correlation matrix of order 70×70.

Consider the matrix multiplication Ru in equation (6) and, in particular, the scalar product of the row j of R_{jk} and the column vector u. This product is

$$R_j u = v_j \Sigma u^2 \tag{7}$$

and this can be written briefly as

$$v_j = \frac{R_j u}{\Sigma u^2} \tag{8}$$

but this is also the well-known equation for the slope of the regression line r on u through the origin for a plot of r against u. If we plot columns of r_{jk} against the trial values u_k, we get a plot with n points. The best fitting straight line through the origin (regression r on u) can be drawn by inspection and the slope of the line is easily read graphically with sufficient accuracy for the first few iterations. That slope is the value v_j. To plot, say, 30 points and to draw by inspection the best fitting straight line through the origin takes less time than to add cumulatively 30 products on a calculating machine.

By equations (5) and (6) it is evident that at the solution the values $v_j = u_j = a_j$. When a set of values of v has been determined graphically, we plot v against u and find the slope m. In the first few graphical iterations, this slope will not be unity because the trial values u are probably too large or too small. If the true values are better represented by ku, in which k is a stretching factor, then a plot of r against ku will give a slope of v/k instead of v. When these graphs have been drawn, the slope of v/k against ku should be unity. But the obtained slope of v against u is m for the values actually used. Hence the observed slope $m = k^2$. Having found the slope m of the plot of v against u, we find $k = \sqrt{m}$. Then if we should take a new set of trial numbers $x_j = ku_j$, the iteration would give, instead of equation (8),

$$\frac{Rx}{\Sigma x^2} = y, \tag{9}$$

where $y = v/k$. The slope of y against x should now be unity and the values of y should be used as the trial vector for the next graphical iteration. The determination of m and k can be done with a slide rule and the new trial values $y = v/k$ can also be determined by the slide rule. The slope m can be found either by inspection or by simple summing with the method of averages in which $m = \Sigma v/\Sigma u$ for like signed pairs of v and u.

The graphical method here described for the principal axes solution has also been adapted for a centroid method of factoring the correlation matrix by which the computations for a factor matrix can be done by a computer

without excessive labor even for a large matrix and without the use of tabulating machine equipment. That procedure is considerably faster and it will be described in a subsequent paper.

A numerical example is here given. In table 1 we have a correlation matrix R of order 10×10 and rank 3. The corresponding factor matrix F for the principal axes is shown in the same table. It is the solution to be found from R. The latent roots of R are 2.85, 1.47 and 0.63. These are the sums of squares of columns of F. In table 2 the initial trial vector u for the major principal factor is taken roughly proportional to the sums of columns of R. Column 1 of R is plotted against column u and the slope is estimated to be approximately $+0.05$ and it is recorded in column v_1. Ten such plots give the values in column v_1. The slope is read directly from each graph by noting the ordinate of a straight line fit at $u_1 = 1$. Next obtain the two column sums Σu_1 and Σv_1. The ratio $\Sigma v/\Sigma u = m$ and $k = \sqrt{m}$. This summation can be absolute sums for like signed pairs of u and v. Then compute $y_1 = v_1/k$ with a slide rule. These are also the trial values u_2 for the next trial. Proceed likewise for three trials which give the desired values of a_{j1} to two decimals.

The first factor residuals are then computed. These are $\rho_{jk} = r_{jk} - a_{j1}a_{k1}$. Choose as a starting vector that column of the first factor residuals which has the largest absolute sum, ignoring diagonals. This is column 1. Its values are recorded in column u_1 for factor two. The procedure is now the same as before. The third iteration was taken on a calculating machine and it gave the second factor loadings a_{j2}, the second column of F, to two decimals. The last iterations for each factor can be done on a calculating machine to obtain greater accuracy while the first few iterations can be done graphically to save time and labor. The computation for the third column of F was done in the same manner.

Occasionally, when two or three latent roots are nearly the same, the iterative process will be found to oscillate or the convergence will be slow. This is an indication that the test configuration has nearly equal thicknesses in these dimensions, so that the configuration is nearly circular or spherical in these dimensions. If the purpose is to extract the maximum variance from the correlation matrix, it does not matter where we place a set of orthogonal axes in those dimensions which are represented by nearly equal latent roots. In such a situation it is desirable to reduce the obtained vector v to a unit vector in the system without waiting for complete convergence. That can be done with a stretching factor p on v so that $pv = a$, where a are the desired factor loadings. The value of p can be found from the relation $1/p = \sqrt{\Sigma uv}$. By this device the computer need not be unduly delayed by slow convergence in obtaining a factorially useful solution.

TABLE 1

	CORRELATION MATRIX R										FACTOR MATRIX F			
	1	2	3	4	5	6	7	8	9	10		I	II	III
1	0.37	0.33	−0.29	0.11	0.10	0.16	−0.33	−0.09	0.20	−0.03	1	0.00	0.60	0.10
2	0.33	0.46	−0.31	0.18	0.22	0.05	−0.15	−0.14	0.20	0.18	2	0.10	0.60	−0.30
3	−0.29	−0.31	0.30	−0.05	−0.04	0.04	0.34	0.27	0.03	0.15	3	0.20	−0.50	0.10
4	0.11	0.18	−0.05	0.14	0.18	0.15	0.11	0.14	0.28	0.30	4	0.30	0.20	−0.10
5	0.10	0.22	−0.04	0.18	0.24	0.16	0.20	0.18	0.34	0.42	5	0.40	0.20	−0.20
6	0.16	0.05	0.04	0.15	0.16	0.45	0.08	0.43	0.54	0.33	6	0.50	0.20	0.40
7	−0.33	−0.15	0.34	0.11	0.20	0.08	0.70	0.43	0.27	0.63	7	0.60	−0.50	−0.30
8	−0.09	−0.14	0.27	0.14	0.18	0.43	0.43	0.62	0.56	0.54	8	0.70	−0.20	0.30
9	0.20	0.20	0.03	0.28	0.34	0.54	0.27	0.56	0.77	0.66	9	0.80	0.30	0.20
10	−0.03	0.18	0.15	0.30	0.42	0.33	0.63	0.54	0.66	0.90	10	0.90	0.00	−0.30
Σ	0.53	1.02	0.44	1.54	2.00	2.39	2.28	2.94	3.85	4.08	Σ	4.50	0.90	−0.10

TABLE 2

	FACTOR I						FACTOR II					
	TRIAL 1		TRIAL 2		TRIAL 3		TRIAL 1		TRIAL 2		TRIAL 3	
	u_1	v_1	$y_1 = u_2$	v_2	$y_2 = u_3$	v_3	u_1	v_1	$y_1 = u_2$	v_2	$y_2 = u_3$	v_3
1	0.10	0.05	0.05	0.10	0.10	0.00	0.37	1.00	0.59	0.60	0.61	0.60
2	0.25	0.15	0.16	0.10	0.10	0.10	0.33	1.00	0.59	0.62	0.63	0.60
3	0.10	0.15	0.16	0.20	0.20	0.20	−0.29	−0.80	−0.47	−0.50	−0.51	−0.50
4	0.40	0.30	0.33	0.30	0.30	0.30	0.11	0.35	0.21	0.15	0.15	0.20
5	0.50	0.40	0.44	0.40	0.40	0.40	0.10	0.35	0.21	0.20	0.20	0.20
6	0.60	0.50	0.55	0.50	0.51	0.50	0.16	0.50	0.30	0.20	0.20	0.20
7	0.60	0.50	0.55	0.60	0.61	0.60	−0.33	−0.90	−0.53	−0.50	−0.51	−0.50
8	0.70	0.60	0.66	0.70	0.71	0.70	−0.09	−0.30	−0.18	−0.20	−0.20	−0.20
9	0.90	0.80	0.87	0.80	0.81	0.80	0.20	0.55	0.33	0.30	0.31	0.30
10	1.00	0.85	0.93	0.90	0.91	0.90	−0.03	0.00	0.00	0.00	0.00	0.00
Σ	5.15	4.30	4.70	4.60			2.01	5.75	3.41	3.27		
m		0.8350		0.9787				2.860		0.959		
k		0.9138		0.9893				1.691		0.979		

The graphical method here described for estimating inner products can be applied in a variety of computational problems.

[1] The author wishes to express his indebtedness to the Carnegie Corporation of New York for a research grant in support of our development of multiple factor analysis and its application to the study of primary mental abilities.

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THE DEVELOPMENT OF NORMAL AND HOMOZYGOUS BRACHY (T/T) MOUSE EMBRYOS IN THE EXTRAEMBRYONIC COELOM OF THE CHICK†

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The study of the causal morphology of mammalian embryos in spite of the great interest attached to it has not progressed very far because of the technical difficulties with which any experimental approach to the problem has met. Waddington and Waterman[1] grew rabbit embryos *in vitro* for a limited time and Nicholas and Rudnick[2] devised a method for raising rat embryos in a culture medium. In spite of these attempts, it was not possible to operate on mammalian embryos in early stages and let them continue their development inside the uterus, nor could such embryos be raised and develop normally in a suitable extra-uterine medium for any length of time. Methods which would accomplish this would be of importance not only for the study of normal causal morphology of mammalian embryos, but also for an experimental study of the embryogeny of certain hereditary abnormalities. Searching for such a method, a new procedure for raising entire mouse embryos outside the uterus was developed and described in detail (Gluecksohn-Schoenheimer).[3] It consisted in removing the embryos from the uterus of the mother and transplanting them into the extra-embryonic coelom of the chick embryo where they remained and developed for one or several days. This method has been applied in the experiments reported here to the study of embryos homozygous for the Brachyury mutation *TT*.

As described by Chesley[4] the homozygous mutants (*T/T*) show extreme morphological abnormalities from the age of about 8 days on and die at about 10 days. The posterior body region, including posterior limb buds, is missing completely and extensive abnormalities are found in notochord, neural tube and somites.

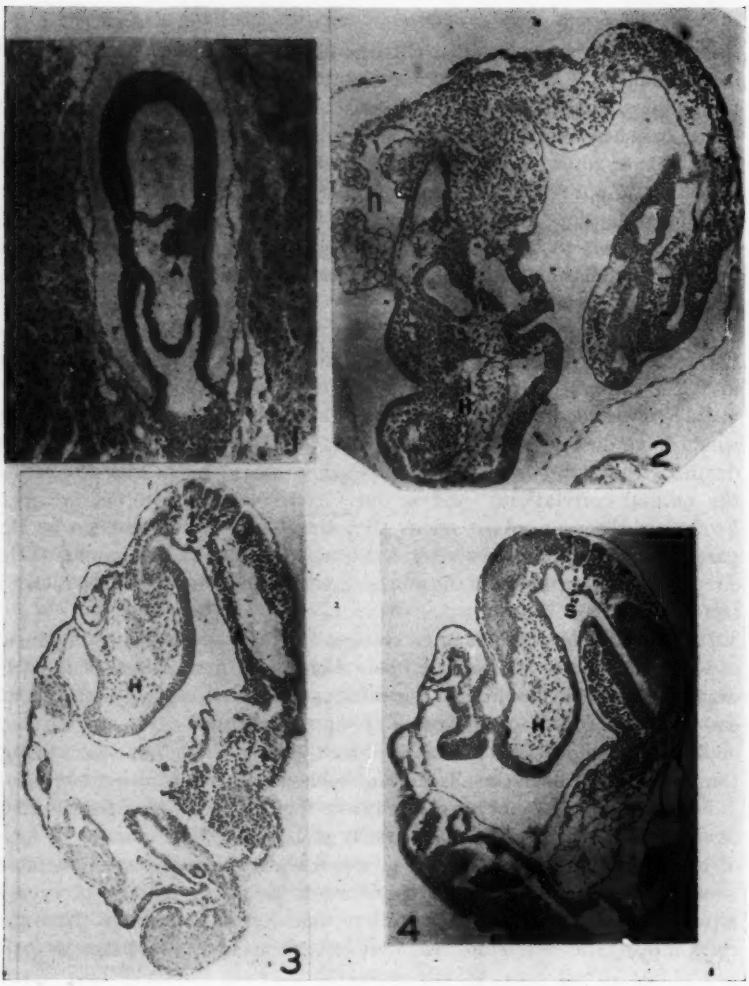
An additional abnormality, not mentioned by Chesley, was observed in the allantois. While the normal allantois, after having budded off from the embryo, appears turgid and proceeds to grow and to lengthen until it comes in contact with the ectoplacental cone, the abnormal allantois of the T/T homozygotes stays very short and has a shrunken tuberous appearance. As a result of the abnormality of the allantois, umbilical vessels do not develop; thus the connection of the embryonic and the maternal circulation fails to be established and the embryo dies at the time when maternal circulation becomes all-important for the nutrition of the embryo (age of 10 days).

Ephrussi,[5] in an attempt to determine the potentialities of the different tissues of the inviable T/T embryos, explanted these tissues and grew them in tissue culture. Under the experimental conditions, the tissues survived, grew and differentiated beyond the time and stage at which they would have died *in utero*. From his results, Ephrussi concludes that the death of the embryos *in utero* is probably caused by a "disturbance of the normal correlations" and is due "exclusively to internal factors." From our observations, it seems that the death of the embryo at the particular time of 10 days after fertilization is due to the failure of the circulatory connection with the mother rather than to any unknown internal factors.

It seemed of considerable interest to study the extra-uterine development of whole T/T embryos from two points of view: first, it was planned to examine their development in the chick host and compare it with that of embryos of the same genotype (T/T) in their normal environment; secondly, it was hoped that by this approach we might be able to learn something about the rôle of the allantois in the development of the homozygotes.

For this purpose, embryos derived from matings of heterozygous Brachy females ($T/+$) to heterozygous Brachy males ($T/+$) were used. A total of 149 transplants was made, 84 of which were successful. A transplant was considered successful if development of the mouse embryo proceeded after transplantation and if the embryo was alive when removed from the chick host. The mouse embryos were left in the chick embryo for 18 to 47 hours. After this time, they were taken out from the chick host, examined, recorded, fixed in Bouin, imbedded in paraffine, sectioned at 6 μ and stained in Delafield's Haematoxylin and eosin. The results to be reported here were arrived at after examination of the sections.

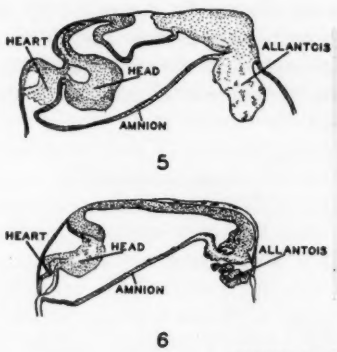
As described before (Gluecksohn-Schoenheimer),[3] normal mouse embryos removed from the uterus of the mother 7 days after copulation (egg cylinder stage, Fig. 1) and transplanted into the extra-embryonic coelom of the chick develop and differentiate head structures, neural folds, somites, a beating heart and a tail bud (Fig. 2). The embryos from matings of Brachy ($T/+$) by Brachy ($T/+$), transplanted into the extra-



FIGURES 1–4

Figure 1. Sagittal section through an egg cylinder stage of a normal mouse embryo, 7 days 19 hours after copulation, = stage 11. A = Allantois. 80×. Figure 2. Sagittal section through a normal mouse embryo (No. 207) explanted into the extra-embryonic coelom of the chick in the egg cylinder stage (stage 11) and removed from there 49 hours later. H = head, h = heart. 80×. Figure 3. Sagittal section through a mouse embryo (No. 314) from a mating of $T/+ \times T/+$, explanted into the extra-embryonic coelom of the chick in the egg cylinder stage (stage 11) and removed from there 24 hours later. Embryo belongs into Class I = normal. H = head, S = somites. 80×. Figure 4. Sagittal section through the same embryo (No. 314). Note turgid attached allantois. H = head, S = somites, A = allantois. 80×.

embryonic coelom of the chick, fall into three classes: the first class comprises those embryos which develop normally in the host environment; they show good development of head, heart, somites and neural tube (Fig. 3); the allantois is big and turgid and attached to the ectoplacental cone (Fig. 4). The rest of the embryos show abnormal development and can, in turn, be separated into two classes, using the condition of the allantois as criterion. In both of these classes, II and III, the allantois is abnormal, but in each class typically different from the other: Class II contains embryos with a short allantois which does not attach to the ecto-



FIGURES 5–7

Figure 5. Sagittal section through mouse embryo (No. 413) from mating of $T/+ \times T/+$, grown in extra-embryonic coelom of the chick; Class II. No somites, underdevelopment of head and trunk. Short allantois. 80×. Drawn from photograph. Figure 6. Sagittal section through mouse embryo (No. 312) from mating of $T/+ \times T/+$, grown in extra-embryonic coelom of chick; Class III. No somites, underdevelopment of head and neural folds; note the short tuberous allantois. 80×. Drawn from photograph. Figure 7. Photograph of a mouse embryo (T/T, Class III) which was explanted into the extra-embryonic coelom of the chick in the early head fold stage (8 days) and remained there for 47 hours. Typical features of T/T embryo: development of head, heart and anterior limb-buds, absence of posterior body structures. Compare Chesley[4], Fig. 6. 20×.

placental cone; but while it is not big and turgid, it is not shrunken either. It looks as though it had been arrested in its development towards normal. The general condition and development of the embryos in this class are not as good as of those in the normal group: head and trunk structures are underdeveloped and somites are sometimes lacking entirely (Fig. 5). Whether there exists any causal relationship between the underdevelopment of the allantois and that of the rest of the embryo, or whether the abnormal allantois presents just one symptom of a general tendency to underdevelopment of the entire embryo cannot be decided now. The embryos of the

second abnormal group, Class III, are characterized by an allantois with a very typical abnormal appearance: it is small, not turgid but collapsed, and characteristically tuberous; it stays short and never attaches to the ectoplacental cone. Its most characteristic feature is its tuberosity (Fig. 6). There is definite underdevelopment in the rest of the embryo; the head is small, the trunk short, the neural folds are thin, there are no somites, and hardly ever a beating heart. Four of the embryos in Class III developed into typical T/T homozygotes (Fig. 7); all of these had been explanted in early head fold stages, i.e., at just 8 days after copulation. The rest of the embryos in this class which had been explanted in earlier stages—i.e., egg cylinder stage—could not be diagnosed by any of the typical features of 9-day T/T homozygotes because they did not develop far enough. But they were diagnosed by the typically abnormal condition of the allantois; and in addition they showed underdevelopment of head and trunk and absence of somites.

TABLE 1

	EMBRYOS OBTAINED				
	CLASS I NORMAL	CLASS II ABNORMAL	CLASS III ABNORMAL T/T	?	TOTAL
Explantation stage of embryos,					
Stage: 7–10[a]	3	4	2		9
Stage: 11–13[b]	8	22	11	5	46
Stage: early head fold, 8 days	17	1	6	2	26
Total	28	27	19	7	81

[a] Early egg cylinder stages, end of 6 days.

[b] Egg cylinder stages, 7 days, beginning growth of allantois, slight thickening of head folds.

When we consider the numerical distribution of the explanted embryos among the three different classes (table 1), we find that 19 out of a total of 81 fall into Class III, i.e., the group comprising the embryos supposed to represent the abnormal TT class. Of these 19, only 4 developed all of the typical TT features; but all of the others had the typically abnormal tuberous allantois which is found in all TT embryos developing in their normal environment (see above) and which never appears in explanted embryos from matings of normal by normal. We expect one quarter of the embryos from $T/+$ by $T/+$ matings to be T/T and the actual number of 19 out of 81 fits expectation very well.

The second class of abnormals—27 embryos—might possibly represent the $T/+$ embryos. If one considers the different explantation stages separately in this class, it appears that of the embryos explanted at earlier stages (7–13) about 50% fall into Class II while of those explanted at later stages (early head fold and 8-days stage) about 70% fall into Class I and only one embryo is classified as Class II. It is conceivable that the

developmental potencies of the very early $T/+$ embryos under the unfavorable explantation conditions are reduced in comparison with those of $+/+$ embryos. If this is the case, it might well be that the allantois, which is completely abnormal in the homozygotes, reacts to the abnormal explantation conditions more strongly in the young heterozygous embryo than it does in the homozygous normal, showing lower developmental potencies under abnormal conditions than does the normal. In this connection it seems of interest that a deficiency of the number of heterozygous short-tailed mice ($T/+$) appeared in matings of Brachy short-tailed mice ($T/+$) *inter se* (Kobozieff and Pomriaskinsky-Kobozieff)[6] and in our own laboratory (Dunn).[7] Although some of this deficiency could be accounted for by the failure of the Brachy mutation to express itself in some of the heterozygotes, a differential prenatal mortality of $T/+$ embryos might be responsible for the lower number of heterozygotes in addition to the normal appearance of some $T/+$ mice. The total number of embryos in Class II does not correspond to the expected 50% of $T/+$ embryos while the number of 28 in the normal group is higher than the expected 25%. One would thus have to assume that a certain fraction of $T/+$ embryos, namely, the older ones, were able to develop normally, or one might ascribe the distribution of embryos among the two classes entirely to chance. In the latter case, one would assume identical developmental potencies for $+/+$ and $T/+$ embryos, and they would be equally distributed between Classes I and II. Their total of 55 corresponds well to the expected 75% of $+/+$ and $T/+$ embryos combined. While, thus, there remain some doubts about ascribing the embryos in Classes I and II to two genetically different groups, $+/+$ and $T/+$, the remaining 25% fall into a group distinctly and characteristically different from the rest of the embryos, independent of the stage of explantation; this group is assumed to represent the T/T class.

It thus appears that the abnormal development of T/T embryos is determined before it is apparent morphologically and is independent of the stage of explantation (table 1). The earliest stages which it is possible to explant, i.e., early egg cylinder stages, show the tendencies to abnormal development of the entire embryo. Earlier stages cannot be tested, since they do not develop at all under the explantation conditions described, regardless of whether they come from crosses of normals or Brachys.

An analysis of the rôle of the allantois in the development of the normal and abnormal embryos was not possible on the basis of our experiments. The fact that an abnormal or underdeveloped allantois occurs always in combination with abnormal or subnormal development of the entire embryo seems highly suggestive. The allantois of the mouse might well serve as an organ for intake of nutrient substances and excretion of waste products. Any disturbance in the function of this organ might thus lead

to secondary abnormalities throughout the entire embryo. However, experiments involving the extirpation of the allantois and studying its effects on the embryo would be required to settle the question of the rôle of the allantois in the development of the normal mouse embryo. Such experiments might also provide an answer to the question of the possible causal rôle of the allantois in the abnormal development of TT embryos.

Summary.—Embryos from crosses of Brachy short-tailed mice ($T/+$) *inter se* were removed from the mother in early stages of development and transplanted to the extra-embryonic coelom of the chick. They remained in the host for 18 to 47 hours and were then removed for study. The embryos obtained could be separated into three different classes: (1) normal embryos; (2) abnormal embryos with different degrees of abnormalities, with reduced developmental potencies, possibly representing the $T/+$ embryos; (3) abnormal embryos with typical abnormalities, representing T/T embryos. T/T embryos will thus develop true to their genotype, even if explanted before the time at which morphological differences appear. The possible causal rôle of the allantois in the development of these embryos is discussed.

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REMARK ON A DISTORTION TENSOR FOR ELASTIC DISPLACEMENTS

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One of the most successful of the various conditions for yield resulting from a state of stress produced by mechanical means is that due to von Mises.[1] This condition states that yield will occur when the energy per unit volume produced by a change of shape alone reaches a certain value characteristic of the material under consideration. It would thus appear

that distortion, i.e., change of shape as distinct from change of volume, is one of the primary factors in the occurrence of yield. This suggests that in addition to the ordinary tensors of stress and strain (related by Hooke's law) we consider a distortion tensor and seek to express the condition of yield directly in terms of this tensor.

A differentiable point transformation $x \rightarrow \bar{x}$ referred to rectangular coordinates and having its jacobian everywhere different from zero (and hence locally non-singular) will be conformal (i.e., angles will be left unaltered) if, and only if,

$$\Sigma \frac{\partial \bar{x}^\alpha}{\partial x^\beta} \frac{\partial \bar{x}^\alpha}{\partial x^\gamma} = \rho \delta_{\beta\gamma}, \qquad (\rho > 0), \tag{1}$$

where ρ is some positive function of position. When the transformation is an elastic displacement it is customarily represented by $\bar{x}^\alpha = x^\alpha + u^\alpha(x)$ in which the u^α (also denoted by u_α since rectangular coördinates are employed) are the components of a vector. In accordance with the usual assumptions, the u's and their derivatives are small quantities, the product of which can be neglected. Hence

$$\frac{\partial \bar{x}^\alpha}{\partial x^\beta} = \delta^\alpha_\beta + u^\alpha_{,\beta}, \tag{2}$$

and

$$\delta_{\beta\gamma} + u_{\beta,\gamma} + u_{\gamma,\beta} = \rho \delta_{\beta\gamma}, \tag{3}$$

when (2) is substituted into (1). Taking the determinant of both members of (1) and making use of the form (2) of the derivatives $\partial \bar{x}^\alpha / \partial x^\beta$ we have

$$\left|\delta^\alpha_\beta + u^\alpha_{,\beta}\right|^2 = \left|\rho \delta_{\alpha\beta}\right| = \rho^3,$$

or

$$\rho = \left|\delta^\alpha_\beta + u^\alpha_{,\beta}\right|^{3/2} = 1 + {}^2/_3 u^\alpha_{,\alpha} = 1 + {}^2/_3 \Delta, \qquad (\Delta = u^\alpha_{,\alpha}).$$

Substitution of this value of ρ into (3) gives

$$e_{\alpha\beta} - \frac{\Delta}{3} \delta_{\alpha\beta} = 0, \qquad (e_{\alpha\beta} = {}^1/_2(u_{\alpha,\beta} + u_{\beta,\alpha})), \tag{4}$$

where e is the strain tensor of elasticity theory. The quantity Δ is the dilatation or increase per unit volume due to the displacement. Conversely if (4) is satisfied the elastic displacement is conformal.

We now define a tensor D as an invariant of an arbitrary elastic displacement by putting

$$D_{\alpha\beta} = e_{\alpha\beta} - \frac{\Delta}{3} \delta_{\alpha\beta}.$$

We call D the *distortion tensor*. From the above, the displacement will be conformal (i.e., without distortion) if, and only if, $D = 0$. We can therefore think of the tensor D, in general, as giving a measure of the distortion produced by the elastic displacement.[2]

It is well known that any elastic displacement causes a displacement of the local neighborhood of an arbitrary point P which is the result of the following three displacements, namely (α) a translation $P \to Q$, (β) a rigid rotation about Q, and (γ) a non-rigid displacement or deformation having Q as a fixed point. This latter deformation can be resolved, on the basis of the distortion tensor D, into a component with distortion and a component without distortion. Thus if δx denotes an arbitrary small vector issuing from the above point Q, the part of the deformation due to the distortion will be represented by $D_{\alpha\beta}\delta x^{\beta}$ and the part without distortion by $(\Delta/3)\delta_{\alpha\beta}\delta x^{\beta}$ these quantities being the components of the changes produced in δx^{α} in this decomposition of the deformation.

Hooke's law for a homogeneous and isotropic medium can now be written

$$E_{\alpha\beta} = 2\mu D_{\alpha\beta}, \qquad \Theta = (3\lambda + 2\mu)\Delta, \tag{5}$$

where

$$E_{\alpha\beta} = \sigma_{\alpha\beta} - \frac{\Theta}{3}\delta_{\alpha\beta}, \qquad \Theta = \Sigma\sigma_{\alpha\alpha}.$$

Thus E is the distortion analogue of the stress tensor σ while λ and μ are the usual elastic constants. Now denote by τ_i and γ_i ($i = 1, 2, 3$) the principal values (at any point) of the tensors σ and E, respectively. From the definition of these quantities we immediately have the relations $\gamma_i = \tau_i - \Theta/3$ and when written in full these become

$$\left.\begin{aligned} \gamma_1 &= {}^1/_3[(\tau_1 - \tau_2) + (\tau_1 - \tau_3)], \\ \gamma_2 &= {}^1/_3[(\tau_2 - \tau_3) - (\tau_1 - \tau_2)], \\ \gamma_3 &= {}^1/_3[-(\tau_1 - \tau_3) - (\tau_2 - \tau_3)]. \end{aligned}\right\} \tag{6}$$

Now consider the square of the tensor E. Denoting this by E^2 we have

$$\begin{aligned} E^2 = \Sigma E_{\alpha\beta}E_{\alpha\beta} &= \Sigma(\gamma_\alpha\delta_{\alpha\beta})(\gamma_\alpha\delta_{\alpha\beta}) = \Sigma\gamma_i\gamma_i \\ &= {}^1/_3[(\tau_1 - \tau_2)^2 + (\tau_1 - \tau_3)^2 + (\tau_2 - \tau_3)^2], \end{aligned}$$

when use is made of the above relations (6). But apart from a constant factor the above bracket expression is the energy per unit volume due to distortion.[3] Since the squares of the tensors D and E differ only by a constant factor on account of (5), the von Mises yield condition can therefore be stated as follows: *yield will occur when the square of the distortion tensor D (or its stress analogue E) attains a characteristic value.*

By a distortion invariant of an elastic displacement we understand any invariant which depends solely on the distortion. In particular any scalar

function of the components $\sigma_{\alpha\beta}$ (or $e_{\alpha\beta}$) will be a distortion invariant if, and only if, it is expressible as a function of the components $E_{\alpha\beta}$ (or $D_{\alpha\beta}$). The sufficiency of this condition is immediately obvious. To show the necessity for a scalar $S(\sigma_{\alpha\beta})$ we have

$$S(\sigma_{\alpha\beta}) = S(\tau_\alpha \delta_{\alpha\beta}) = S[(\tau_\alpha + \tau)\delta_{\alpha\beta}]$$

where τ is arbitrary. But choosing $\tau = -\Theta/3$ the latter function is seen to become $S(\gamma_\alpha \delta_{\alpha\beta})$or $S(E_{\alpha\beta})$ from which the conclusion follows. This result can obviously be extended to differential and non-scalar invariants. The above quantity E^2 or D^2 is an example of a distortion scalar. So likewise is the quantity $\gamma_1 - \gamma_3 = \tau_1 - \tau_3$ which is proportional to the maximum shearing stress (when $\tau_1 \geqq \tau_2 \geqq \tau_3$). This stress also appears to be of significance in determining the condition of yield.[4] Other examples are the magnitudes of the principal values γ_i of the tensor E (which differ from the principal values of the distortion tensor by a constant factor). Useful modifications of the known yield conditions may possibly be given by these latter or other distortion scalars which can be constructed. Such invariants will automatically take care of the failure to reach a state of yield as the result of hydrostatic pressure or uniform tension.

[1] *Nachr. d. Gesellsch. d. Wissensch. zu Göttingen, Math.-Phys. Klasse*, pp. 582–592 (1913). See also, Nadai, A., *Plasticity*, McGraw-Hill, Chap. 13 (1931), and Timoshenko, S., *Theory of Elasticity*, McGraw-Hill (1934), p. 137.

[2] The usual formula for the change $\delta\theta$ of an angle θ due to an elastic displacement gives this change in terms of the components $e_{\alpha\beta}$ of the strain tensor. Cf. Love, *The Mathematical Theory of Elasticity*, Cambridge, Vol. 4 (1927), p. 62. Also, Timoshenko, S., loc. cit., p. 192. By making use of the above definition of the components $D\alpha\beta$ it can be shown that $\delta\theta$ is given by

$$\sin\theta\, \delta\theta = (D_1 + D_2)\cos\theta - 2D_{\alpha\beta}\nu_1^\alpha \nu_2^\beta$$

where,

$$D_1 = D_{\alpha\beta}\nu_1^\alpha \nu_1^\beta, \qquad D_2 = D_{\alpha\beta}\nu_2^\alpha \nu_2^\beta,$$

and ν_1 and ν_2 are unit vectors determining the angle θ. Hence the change in an angle produced by an elastic displacement is expressible directly in terms of the distortion tensor D.

[3] See Nadai, S., loc. cit., p. 72, and Timoshenko, S., loc. cit., p. 137.

[4] See, for example, Nadai, A., loc. cit., p. 60.

THE MINIMUM RANK OF A CORRELATION MATRIX

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A correlation matrix is a symmetric matrix R with prescribed non-diagonal elements (correlation coefficients) and the matrix problem with which we are concerned is that of determining the diagonal elements (communalities) of R so that the resulting R will have minimum rank ρ. In a recent article[1] I defined the *ideal rank* r of R to be the largest order of a non-vanishing minor of R such that all elements of the minor are correlation coefficients. Evidently $\rho \geqq r$ and I gave a procedure for determining the communalities under the hypothesis that $\rho = r$.

Indeed we may always select our notation for the elements of M so that

$$R = \begin{pmatrix} A & B' & G' \\ B & C & H' \\ G & H & K \end{pmatrix},$$

where A, B, C are r-rowed square matrices and the matrix B has an inverse $B^{-1} = E$. Then I proved the

LEMMA 1: *If the ideal rank of R is r the matrix $K - GEH'$ is a diagonal matrix. The rank of R is r if and only if $K = GEH'$, $H = GEC$, $G = HE'A$, $B' = AEC$.*

In cases usually encountered in factor analysis the ideal rank r is much less than the order n of R and the r-columned matrices G and H normally have their *maximum* rank r. The purpose of the present note is to provide a proof of the rather astonishing

THEOREM. *Let the ideal rank r of R be also the rank of its submatrices G and H. Then there exist unique communalities such that the resulting correlation matrix R has rank r.*

We shall prove this result by several applications of the first statement in Lemma 1. Since K is symmetric it implies that GEH' is symmetric. Let the rank of G be r so that we may select a notation for R so that

$$G = \begin{pmatrix} S \\ P \end{pmatrix}, \qquad H = \begin{pmatrix} T \\ Q \end{pmatrix}$$

for r-rowed square matrices S and T such that S is non-singular. Define a non-singular matrix

$$L = \begin{pmatrix} I & 0 \\ -PS^{-1} & I \end{pmatrix}$$

of $n - 2r$ rows, for appropriately selected identity matrices I, and form the products

$$LG = \begin{pmatrix} S \\ 0 \end{pmatrix}, \qquad LH = \begin{pmatrix} T \\ W \end{pmatrix},$$

where $W = Q - S^{-1}PT$. Then

$$L(GEH')L' = (LG)E(LH)' = \begin{pmatrix} SE \\ 0 \end{pmatrix}(T'W') = \begin{pmatrix} SET' & SEW' \\ 0 & 0 \end{pmatrix}$$

is a symmetric matrix and necessarily $SEW' = 0$. Since SE is non-singular we have proved that $W' = 0$, $W = 0$,

$$LH = \begin{pmatrix} T \\ 0 \end{pmatrix}, \quad L(GEH')L' = L\begin{pmatrix} SET' & SEQ' \\ PET' & PEQ' \end{pmatrix}L' = \begin{pmatrix} SET' & 0 \\ 0 & 0 \end{pmatrix}.$$

Suppose now that H has rank r so that LH has rank r and T must have rank r. Then T is non-singular and we may prove

LEMMA 2: *The matrices $A - B'T^{-1}S$ and $C - BS^{-1}T$ are diagonal matrices.*

For the submatrix of R consisting of its first $3r$ rows and columns is a correlation matrix

$$N = \begin{pmatrix} A & B' & S' \\ B & C & T' \\ S & T & U \end{pmatrix},$$

which is exactly like R except that K has been replaced by U, G by S, H by T. Every diagonal element of N is a diagonal element of M and B is an r-rowed non-zero minor of N. Then the ideal rank of N is r and, by the first part of Lemma 1, $U - SB^{-1}T'$ is a diagonal matrix. However, this result is actually a part of the statement that $K - GEH'$ is diagonal. We now obtain two correlation matrices

$$N_1 = \begin{pmatrix} U & T & S \\ T' & C & B \\ S' & B' & A \end{pmatrix}, \qquad N_2 = \begin{pmatrix} A & S' & B' \\ S & U & T \\ B & T' & C \end{pmatrix}$$

of ideal rank r from N. The matrix N_1 is obtained by interchanging the first and third blocks of r rows and columns in N and the matrix N_2 by interchanging the second and third blocks of r rows and columns in N. Our lemma then follows from the first part of Lemma 1.

If R has rank r then N, N_1, N_2 have rank r. Applying Lemma 1 we have the uniqueness part of our theorem which we state as

LEMMA 3: *Let R have rank r. Then the communalities are uniquely determined by the equations*

$$A = B'T^{-1}S, \qquad C = BS^{-1}T, \qquad K = GB^{-1}H'. \tag{1}$$

This lemma provides a modification of my earlier technique for computing the communalities. In the new technique we compute the inverses of the *three* matrices S, T, B and the diagonal elements of three matrix products.

To complete the proof of our theorem we assume now that R has ideal rank r and that r is the rank of G and H. By Lemmas 1 and 2 we may select the diagonal elements of R so that (1) is satisfied. Then

$$L(H - GEC) = \begin{pmatrix} T \\ 0 \end{pmatrix} - \begin{pmatrix} SEC \\ 0 \end{pmatrix} = \begin{pmatrix} T - SEC \\ 0 \end{pmatrix} = 0$$

since $C = BS^{-1}T$, $T = SEC$. Similarly

$$L(G - HE'A) = \begin{pmatrix} S \\ 0 \end{pmatrix} - \begin{pmatrix} TE'A \\ 0 \end{pmatrix} = \begin{pmatrix} S - TE'A \\ 0 \end{pmatrix} = 0$$

since $A = B'T^{-1}S$, $S = TE'A$. But L is non-singular and therefore $H = GEC$, $G = HE'A$. Finally, we have the identity

$$GE(B' - AEC)' = (H - GEC)E'A + G - HE'A$$

so that, since the right member is zero, $GE(B - AEC)' = 0$. The first r rows of this product form the matrix $SE(B' - AEC)' = 0$. Thus $(B' - AEC)' = 0$, $B' = AEC$, the rank of R is r. This completes the proof of our theorem.

Let us close by observing that R cannot have its ideal rank as its rank unless the ranks of G and of H are the same. For this is a consequence of the fact that when $H = GEC$ the rank of H is at most the rank of G, and that when $G = HE'A$ the rank of G is at most the rank of H. However, even when G and H have equal ranks *less than* r the minimum rank of R need not be its ideal rank r. Indeed let $G = H = 0$, K be a diagonal matrix and

$$B = \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}, \qquad A = \begin{pmatrix} x_1 & 0 \\ 0 & x_2 \end{pmatrix}, \qquad C = \begin{pmatrix} y_1 & 1 \\ 1 & y_2 \end{pmatrix}.$$

Then every minor of R with more than two rows and containing no communality has a zero row and so $r = 2$. But $B' = AEC$ now becomes $C = A^{-1}$ and this is false since A^{-1} is a diagonal matrix and C is not a diagonal matrix.

[1] The matrices of factor analysis, *Proc. Nat. Acad. Sci.*, **30**, 90–95 (1944).